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L10 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 5
AN 1998:43246 CAPLUS
DN 128:149718
TI Differential expression of matrix metalloproteinases and their tissue inhibitors in leiomyomata: a mechanism for gonadotropin releasing hormone agonist-induced tumor regression
AU Dou, Qingchuan; Tarnuzzer, Roy W.; Williams, R. Stan; Schultz, Gregory S.; **Chegini, Nasser**
CS Department Obstetrics Gynecology, College Medicine, University Florida, Gainesville, FL, 32610, USA
SO Molecular Human Reproduction (1997), 3(11), 1005-1014
CODEN: MHREFD; ISSN: 1360-9947
PB Oxford University Press
DT Journal
LA English
CC 2-4 (Mammalian Hormones)
Section cross-reference(s): 14
AB Tissue remodelling involving extracellular matrix (ECM) turnover plays a major role in leiomyoma growth and regression, regulated by the combined action of matrix metalloproteinases (MMPs) and the tissue inhibitors of MMPs (TIMPs). The authors postulated that leiomyomata express MMP and TIMP mRNA and protein, and their expression is inversely regulated during tumor growth and gonadotropin-releasing hormone agonist (GnRHa)-induced regression. The authors therefore examd. the expression of mRNA and protein for MMPs (interstitial collagenase, MMP-1; gelatinases, MMP-2 and MMP-9; and stromelysin, MMP-3) and TIMPs (**TIMP-1** and TIMP-2) in leiomyoma and matched unaffected myometrium from GnRHa (lupron)-treated and untreated patients. Reverse transcription-polymerase chain reaction (RT-PCR) and restriction enzyme anal. revealed that leiomyomata and myometrium expressed MMP-1, -2, -3, and -9, as well as **TIMP-1** and -2 mRNA. Quant. RT-PCR indicated that leiomyomata and myometrium during the secretory phase of the menstrual cycle expressed higher levels of MMP and TIMP mRNA compared to the proliferative phase, with low to undetectable levels of MMP-1, -2 and -3 mRNA in the tumors. GnRHa therapy induced an overall redn. in MMP and TIMP mRNA expression in both leiomyomata and myometrium, but a significant decrease in **TIMP-1**, and an increase in MMP mRNA expression compared with untreated tumors. Immunohistochem., MMP-1, -2, -3 and -9 and **TIMP-1** and -2 proteins were localized in leiomyomata and myometrial smooth muscle cells, arteriole wall and connective tissue fibroblasts, with an overall increase in myometrial smooth muscle cells, arteriole wall and connective tissue fibroblasts, with an overall increase in MMP and a decrease in TIMP staining intensity in GnRHa-treated groups. Apparently, MMP and TIMP expression in leiomyoma and myometrium are hormonally regulated, and GnRHa-induced tumor regression is accompanied by an increase in MMP expression with a concomitant disease in **TIMP-1** expression, which may potentially provide an environment favoring ECM degrdn.
ST lupron leiomyoma regression matrix metalloproteinase
IT Artery
(arteriole, wall; differential expression of matrix metalloproteinases and tissue inhibitors in human leiomyomata in relation to a mechanism for gonadotropin-releasing hormone agonist-induced tumor regression)
IT Fibroblast

L10 ANSWER 4 OF 6 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.DUPLICATE
AN 1999:29470320 BIOTECHNO
TI Regulation of matrix metalloproteinases (MMPs) and their tissue inhibitors in human myometrial smooth muscle cells by TGF-.beta.1
AU Ma C.; **Chegini N.**
CS N. Chegini, Dept. of Obstetrics and Gynecology, University of Florida, Gainesville, FL 32610, United States.
SO Molecular Human Reproduction, (1999), 5/10 (950-954), 26 reference(s)
CODEN: MHREFD ISSN: 1360-9947
DT Journal; Article
CY United Kingdom
LA English
SL English
AB The objective of the present study was to determine whether transforming growth factor .beta. (TGF-.beta.) regulates the expression of matrix metalloproteinases (MMP) and the tissue inhibitor of MMP (TIMP) in myometrial smooth muscle cells. Using primary cultures of human myometrial smooth muscle cells we found that these cells express MMP-1, MMP-3, **TIMP-1** and TIMP-2 mRNA and protein, with significantly higher values of TIMP than MMR. We also found that TGF-.beta.1 (1 ng/ml) increased the expression of **TIMP-1** mRNA, while it reduced the expression of MMP-1 and MMP-3 mRNA, compared with untreated controls. In addition, TGF-.beta.1 slightly increased the production of **TIMP-1**, but not TIMP-2. Production of MMP-1 and MMP-3 was reduced by treatment with TGF-.beta.1, compared with the untreated control. A major portion of MMP-1 released into the culture-conditioned media was in complex with **TIMP-1**, and the levels of this complex were reduced by treatment With TGF-.beta.1. In conclusion, the data indicate that myometrial smooth muscle cells express MMP and TIMP mRNA and protein, and their expression is differentially regulated by TGF-.beta.1. Such a differential regulation of MMP and TIMP by TGF-.beta. may influence the rate of extracellular matrix (ECM) turnover following tissue injury, induced during myomectomy and Caesarean section, or in leiomyomas during growth.
CT *matrix metalloproteinase; *tissue inhibitor of metalloproteinase; *transforming growth factor beta1; *gene expression regulation; *myometrium; collagenase; stromelysin; tissue inhibitor of metalloproteinase 1; tissue inhibitor of metalloproteinase 2; messenger RNA; extracellular matrix; tissue injury; myomectomy; cesarean section; leiomyoma; tumor growth; human; female; controlled study; human tissue; human cell; article; priority journal
RN (tissue inhibitor of metalloproteinase) 97837-28-0; (collagenase) 9001-12-1; (stromelysin) 79955-99-0; (tissue inhibitor of metalloproteinase 1) 140208-24-8; (tissue inhibitor of metalloproteinase 2) 124861-55-8

L10 ANSWER 5 OF 6 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.DUPLICATE
AN 1999:30027570 BIOTECHNO
TI Expression of matrix metalloproteinases and tissue inhibitor of matrix metalloproteinases in mesothelial cells and their regulation by transforming growth factor-.beta.1
AU Ma C.; Tarnuzzer R.W.; **Chegini N.**
CS Dr. N. Chegini, Department of Obstetrics/Gynecology, Univ. of Florida College of Medicine, Box 100294, Gainesville, FL 32610-0294, United States.
E-mail: cheginin@obgyn.med.ufl.edu
SO Wound Repair and Regeneration, (1999), 7/6 (477-485), 34 reference(s)
CODEN: WREREU ISSN: 1067-1927
DT Journal; Article
CY United States
LA English
SL English
AB Tissue injury and pelvic inflammation often results in peritoneal scar

tissue formation. The objective of this study was to determine whether mesothelial cells which line the peritoneal cavity express matrix metalloproteinases (MMPs) and tissue inhibitors of matrix metalloproteinases (TIMPs), and if their expression is regulated by transforming growth factor- .beta.1, a key regulator of tissue fibrosis. For this purpose we used Met-5A cells, a cell line derived from human normal mesothelial cells, and for comparative analysis we used U-937 cells, a human monocytic/macrophage cell line. The cells were treated with transforming growth factor-.beta.1 (1 ng/ml) for various time periods and the levels of MMP and TIMP mRNA and protein expression were determined using quantitative reverse transcription- polymerase chain reaction and enzyme-linked immunosorbent assay, respectively. The results indicate that the mesothelial cells and macrophages express MMP-1 (collagenase-1), MMP-3 (stromelysin-**1**), **TIMP-1** and TIMP-2 mRNA and protein at various levels, with significantly higher TIMPs than MMPs, and higher MMP-1 than MMP-3 ($p < 0.001$). The mesothelial cells express significantly less MMP-1, higher MMP3 and similar levels of TIMP in RNA compared to macrophages. In a time-dependent manner, treatment of the mesothelial cells with transforming growth factor-.beta.1 resulted in a significant decrease in the expression of MMP-1, while increasing the expression of **TIMP-1** mRNA ($p = 0.05$). In contrast, MMP-3 and TIMP-2 expression was unaffected in mesothelial cells and in macrophages, compared to untreated controls. There was a significant increase in secreted MMP-1 and TIMP-2 by mesothelial cells following transforming growth factor-.beta.1 treatments in a time-dependent manner ($p = 0.05$ and $p=0.01$), without affecting the secretion of these proteins by macrophages. A major portion of MMP-1 in the culture conditioned media of both cell types was found in complex with **TIMP-1**. The ratios of MMP-**1**/**TIMPs** production were significantly higher than MMP-3/TIMPs in mesothelial cells and macrophages, and progressively decreased following transforming growth factor-.beta.1 treatments ($p<0.05$). In conclusion, these results indicate that mesothelial cells express MMP and TIMP mRNA and protein, and their expression is differentially regulated by transforming growth factor-.beta.1, a mechanism that in part may influence the outcome of peritoneal tissue repair and adhesion formation.

CT *matrix metalloproteinase; *tissue inhibitor of metalloproteinase 1;

ANSWER 6 OF 7 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 5

AN 2000:241573 CAPLUS

DN 132:246382

TI Anti-TIMP-1 antibodies and TIMP-1 antisense oligonucleotides for the prevention of adhesions, and diagnostic method

IN Chegini, Nasser; Burns, James; Diamond, Michael; Holmdahl, Lena

PA Genzyme Corporation, USA; University of Florida

SO PCT Int. Appl., 19 pp.
CODEN: PIXXD2

DT Patent

LA English

IC ICM C12Q001-68
ICS G01N033-53; C07K016-44

CC 1-12 (Pharmacology)
Section cross-reference(s): 9, 15, 63

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000020642	A1	20000413	WO 1999-US23014	19991001
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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	AU 9962859	A1	20000426	AU 1999-62859	19991001
	EP 1117835	A1	20010725	EP 1999-950135	19991001
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

ANSWER 5 OF 7 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.DUPLICATE

AN 2001:33116388 BIOTECHNO

TI Matrix metalloproteinase (MMP-1) and tissue inhibitor of MMP in peritoneal fluids and sera and correlation with peritoneal adhesions

AU Chegini N.; Kotseos K.; Bennett B.; Diamond M.P.; Holmdahl L.; Burns J.

CS Dr. N. Chegini, University of Florida, Department of Obstetrics, Box 100294, Gainesville, FL 32610-0294, United States.
E-mail: cheginin@obgyn.ufl.edu

SO Fertility and Sterility, (2001), 76/6 (1207-1211), 34 reference(s)
CODEN: FESTAS ISSN: 0015-0282

PUI S0015028201028746

DT Journal; Article

CY United States

LA English

SL English

AB Objective: To assess the presence of matrix metalloproteinase (MMP-1) and tissue inhibitor of MMP (**TIMP-1**) in peritoneal fluid and serum of subjects with and without **adhesions**. Design: Cross-sectional study. Setting: Academic research centers. Patient(s): Sixty-three patients who underwent abdominal/pelvic **surgery**. Intervention(s): MMP-**1**, **TIMP-1**, and MMP-**1**-**TIMP-1** complex content. Main Outcome Measure(s): ELISA. Result(s): Peritoneal fluids (PF) and sera of subjects with and without peritoneal **adhesions** contain MMP-**1**, **TIMP-1**, and MMP-**1**-**TIMP-1** complex at varying levels with 10- to 100-fold higher **TIMP**-**1** than MMP-1. Compared with serum, PF contains a lower level of MMP-1 in subjects with mild **adhesions** and without **adhesions**, higher **TIMP-1** in subjects with extensive **adhesions**, and lower MMP-**1**-**TIMP**-**1** complex in subjects with moderate **adhesions**. However, the serum and PF content of MMP-**1**, **TIMP-1**, and MMP-**1**-**TIMP-1** complex was not statistically different among subjects with or without **adhesions**, with the exception of **TIMP-1** in PF of subjects with extensive **adhesions**. MMP1-**TIMP-1** ratio indicates that a major portion of MMP-1 is in complex with **TIMP**-**1**. There was no age- or gender-dependent difference in MMP-1 and **TIMP-1** content in serum or PF. Conclusion(s): Despite differences in MMP-1 and **TIMP-1** levels in serum and PF of subjects with extensive and moderate **adhesions**, there is no correlation between MMP-1 and **TIMP-1**, with the exception of higher **TIMP-1** in PF of subjects with extensive **adhesions**. .COPYRGT. 2001 by American Society for Reproductive Medicine.

CT *appendectomy; *hysterectomy; *cystectomy; *uterine tube ligation; *cesarean section; *peritoneum adhesion; *interstitial collagenase; *tissue inhibitor of metalloproteinase 1; serum; peritoneal fluid; disease association; enzyme linked immunosorbent assay; blood level; protein blood level; disease severity; protein content; age; sex difference; human; male; female; major clinical study; controlled study;

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PubMed Nucleotide Protein Genome Structure PMC Taxonomy OMIM Books

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1: Kidney Int. 2003 Aug;64(2):459-67. Related Articles, Links

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STAT proteins mediate angiotensin II-induced production of TIMP-1 in human proximal tubular epithelial cells.

Chen X, Wang J, Zhou F, Wang X, Feng Z.

Department of Nephrology, Chinese General Hospital of PLA, Beijing, China.

STAT proteins mediate angiotensin II-induced production of TIMP-1 in human proximal tubular epithelial cells. BACKGROUND: Angiotensin II and tissue type inhibitor metalloproteinase-1 (TIMP-1) have been implicated in renal tubulointerstitial fibrosis, but the exact mediating signaling pathway is still unknown. Angiotensin II has been reported to activate signal transducers and activators of transcription (STAT) and induce proliferation of myocyte and vascular smooth muscle cells (VSMC). We hypothesized that the STAT signal pathway is involved in the process of renal tubulointerstitial fibrosis. Therefore, we designed the present study to explore whether angiotensin II could induce TIMP-1 expression in human proximal tubular epithelial cells, and whether it was mediated through the STAT signaling pathway. METHODS: Electrophoretic mobility shift assay (EMSA) was employed to determine the DNA-STAT binding activity. Supershift assay was used to test the components of activated STAT proteins. Nuclear translocation of activated STATs was observed with laser scanning confocal microscopy. TIMP-1 expression was analyzed with Northern and Western blots. Valsartan and PD123319 were used to block the effects of angiotensin II type 1 (AT1) and angiotensin II type 2 (AT2) receptors of angiotensin II, respectively. RESULTS: Cultured human proximal tubular epithelial cells constitutively expressed TIMP-1. Angiotensin II induced TIMP-1, mRNA, and protein expressions in time- and dose-dependent manners, which could be inhibited by the AT1 receptor antagonist valsartan, but not by the AT2 antagonist PD123319. Angiotensin II also activated STAT-DNA binding activity in both dose-dependent and biphasic time-dependent manners, and increased the phosphorylation and nuclear translocation of STAT proteins. To examine the role of STAT in angiotensin II-induced TIMP-1 expression, STAT1 and STAT3 antisense oligonucleotides were used. Northern and Western blots showed that STAT1 and STAT3 antisense oligonucleotides could inhibit angiotensin II-induced TIMP-1 expressions, and STAT1 and STAT3 proteins, respectively, could be supershifted by their polyclonal antibodies. CONCLUSION: STAT1 and STAT3 may, at least in part, mediate angiotensin II-induced TIMP-1 mRNA expression in human renal proximal tubular epithelial cells, implicating a role of

WEST Search History

DATE: Tuesday, July 15, 2003

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L14	6211217.pn.	2	L14
L13	6011017.pn.	2	L13
L12	4886787.pn.	2	L12
L11	6011017.pn.	2	L11
L10	L9 same (vivo or administ$ or therap$ or inject$)	3	L10
L9	L2 adj (anti or antibod$)	13	L9
L8	burns-james.in.	6	L8
L7	diamond-Michael.in.	2	L7
L6	Chegini-Nasser.in.	2	L6
L5	Chegini-Nassar.in.	0	L5
L4	L3 same surg$	2	L4
L3	L2 same adhesion	23	L3
L2	TIMP adj 1	421	L2
DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ			
L1	TIMP adj 1	186	L1

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L2             60 S E1 OR E2 OR E4 OR E5
                E BUNS JAMES?/AU
L3              4 S E1 OR E2 OR E4
L4          17048 S TIMP (A) 1
L5            396 S ADHESION? (S) L4
L6              0 S L4 (S) (L1 OR L2 OR L3)
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L9             11 S L4 AND (L1 OR L2 OR L3)
L10             6 DUP REM L9 (5 DUPLICATES REMOVED)
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